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THE HEALTH PROFESSIONAL'S GUIDE TO

Nutrition Management of Thyroid Disease

NICOLE ANZIANI, MS, RD, CDN, CDCES

With Introduction and Medical Review by
FRANCESCO CELI, MD, MHSC

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Academy of Nutrition and Dietetics
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Chicago, IL 60606

The Health Professional's Guide to Nutrition Management of Thyroid Disease

978-0-88091-019-4 (print)
978-0-88091-022-4 (eBook)
Catalog Number 019420 (print)
Catalog Number 019420e (eBook)

10 9 8 7 6 5 4 3 2 1

For more information on the Academy of Nutrition and Dietetics, visit www.eatright.org.

Library of Congress Cataloging-in-Publication Data

Names: Anziani, Nicole, author.
Title: The health professional's guide to nutrition management of thyroid disease / Nicole Anziani, MS, RD, CDN, CDE ; with introduction and medical review by Francesco Celi, MD, MHSC.
Description: Chicago, IL : Academy of Nutrition and Dietetics, [2020] | Includes bibliographical references and index. |
Summary: "This comprehensive reference provides guidance for dietitians, physicians, and other allied care professionals working with patients with thyroid disease. Using the most current evidence based guidelines from the American Thyroid Association, the American Association of Clinical Endocrinologists, and available research, this guide addresses: Thyroid function, weight regulation, and the role of iodine Energy and nutrient requirements in thyroid disease Diagnosis, medical management, and applying the Nutrition Care Process to provide patient centered nutrition care of hypothyroidism, hyperthyroidism, and thyroid cancer Awareness of additional interventions, including alternative treatments Working with special populations, related conditions and issues, and prevention strategies Useful appendix materials include sample food plans, low iodine diet guidelines, list of goitrogens, monitoring recommendations, suggested resources, and more"--Provided by publisher.
Identifiers: LCCN 2020003212 (print) | LCCN 2020003213 (ebook) | ISBN 9780880910194 (paperback) | ISBN 9780880910224 (ebook)
Subjects: LCSH: Thyroid gland--Diseases--Diet therapy.
Classification: LCC RC655 .A58 2020 (print) | LCC RC655 (ebook) | DDC 616.4/40654--dc23
LC record available at https://lccn.loc.gov/2020003212
LC ebook record available at https://lccn.loc.gov/2020003213

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Frequently Used Terms and Abbreviations

AACE	American Association of Clinical Endocrinologists
AAFP	American Academy of Family Physicians
ACE	angiotensin converting enzyme
ACTH	adrenocorticotropic hormone
ADH	antidiuretic hormone
AI	Adequate Intake
AICR	American Institute for Cancer Research
AITD	autoimmune thyroiditis
AJCC	American Joint Committee on Cancer
AN	anorexia nervosa
ASPEN	American Society for Parenteral and Enteral Nutrition
ATA	American Thyroid Association
ATD	antithyroid drugs
BEE	basal energy expenditure
BIA	bioelectrical impedance analysis
BMI	body mass index
BN	bulimia nervosa
BPA	bisphenol-A
CEA	carcinoembryonic antigen
CH	congenital hypothyroidism
CI EBNPG	Critical Illness Evidence-Based Nutrition Practice Guidelines
CLA	conjugated linoleic acid
CLT	chronic lymphocytic thyroiditis
CPK	creatine phosphokinase
CRP	C-reactive protein test
D3	type 3 iodothyronine deiodinase
DEXA	dual energy x-ray absorptiometry
DKA	diabetic ketoacidosis
DRIs	Dietary Reference Intakes
DTC	differentiated thyroid cancer
DTE	desiccated thyroid extract
DV	Daily Value

EDNOS	eating disorder not otherwise specified
EGCG	epigallocatechin gallate
FDA	US Food and Drug Administration
FNA	fine needle aspiration (biopsy)
FODMAPs	fermentable oligosaccharides, disaccharides , and monosaccharides and polyols
FSH	follicle-stimulating hormone
FT3	free triiodothyronine (T3)
FT4	free thyroxine (T4)
GD	Graves disease
GFR	glomerular filtration rate
GH	growth hormone
GI	gastrointestinal
GO	Graves ophthalmopathy
H/H	hemoglobin/hematocrit
HBE	Harris-Benedict equation
hCG	human chorionic gonadotropin
HCTZ	hydrochlorothiazide
HIIT	high-intensity interval training
HPA	hypothalamic-pituitary-adrenal
HPT	hypothalamic-pituitary-thyroid
HRT	hormone replacement therapy
HSL	hormone-sensitive lipase
IBS	irritable bowel syndrome
IgE	immunoglobulin E
IgG	immunoglobulin G
IIH	iodine-induced hyperthyroidism
LBM	lean body mass
LC-MS/MS	liquid chromatography–tandem mass spectrometry
LDN	low-dose naltrexone
LH	luteinizing hormone
LLLT	low-level laser therapy
LPL	lipoprotein lipase
L-T3	liothyronine
L-T4	levothyroxine
MCTs	medium-chain triglycerides
MCV	mean corpuscular volume
MI	myo-inositol
MMA	methylmalonic acid
MMI	methimazole

MMP	matrix metalloproteinase
MNT	medical nutrition therapy
MTC	medullary thyroid cancer/carcinoma
MTHFR	methylenetetrahydrofolate reductase
NAC	N-acetylcysteine
NCP	Nutrition Care Process
NIH	National Institutes of Health
OH	overt hypothyroidism
PCOS	polycystic ovary syndrome
PES	problem, etiology, signs and symptoms statements
PFASs	perfluoroalkyl and polyfluoroalkyl substances
PTH	parathyroid hormone
PTU	propylthiouracil
RAI	radioactive iodine
RCT	randomized controlled trial
RDA	Recommended Dietary Allowance
RDW	red blood cell distribution width
rhTSH	recombinant human TSH
RMR	resting metabolic rate
RT3	reverse triiodothyronine (T3)
RxWBS	post-treatment whole body scan
SCH	subclinical hypothyroidism
SH	subclinical hyperthyroidism
SHBG	sex hormone binding globulin
SIBO	small intestinal bacterial overgrowth
SSKI	potassium iodide
T1	monoiodothyronine
T2	diiodothyronine
T3	triiodothyronine
T4	thyroxine
TA	toxic adenoma
TAb	thyroid antibody
TBG	thyroxine-binding globulin
TBII	thyrotropin-binding inhibitory immunoglobulin
TEE	total energy expenditure
TFT	thyroid function test
Tg	thyroglobulin
TgAb	thyroglobulin antibody
TIBC	total iron binding capacity
TMNG	toxic multinodular goiter

TPO	thyroid peroxidase/thyroperoxidase
TPOAb	thyroid peroxidase antibody
TRAb	thyroid stimulating hormone receptor antibody
TRH	thyrotropin-releasing hormone
TSH	thyroid-stimulating hormone
TSI	thyroid stimulating immunoglobulin
TT	total thyroidectomy
TT3	total triiodothyronine (T3)
TT4	total thyroxine (T4)
TTR	transthyretin
UBW	usual body weight
UICC	Union for International Cancer Control
UL	Tolerable Upper Intake Level
US	ultrasound
WBS	whole body scan

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Preface

This book is intended mainly for the health care professional and, in that vein, is written using scientific and medical language. I would ask health professionals using this book to approach the nutrition care of thyroid patients with an open mind. Thyroid care is nuanced—no two patients are the same, and each person will present with a different set of clinical parameters and reported symptoms. This book, which contains a wealth of available evidence-based practices and potential interventions at the time of writing, provides guidance for health care practitioners such as physicians, dietitians, and all other allied care professionals working with patients with thyroid disease. My hope is that once a patient has tried "everything" yet still doesn't feel "right," this book will help us as providers to determine additional options to better help them. This book is intended as both a time-saver and a guide, matching nutrition care with medical care, but with the understanding that research will continue to refine and improve recommendations over time.

Throughout the writing and revision phases, I have tried to provide a balanced account of the available research, citing evidence-based guidelines from the American Thyroid Association (ATA) and the American Association of Clinical Endocrinologists (AACE), as well as citing numerous other research studies. I have based the framework of nutrition care on the Academy of Nutrition and Dietetics Nutrition Care Process (NCP). You will also find information from several evidence-based integrative and functional medicine resources included within. Peer reviewers weighed in to help ensure that content was relevant, up-to-date, and useful for practitioners.

Although evidence will continue to evolve from ongoing research, we must remember that our patients are people, not study subjects. There may not yet be robust research to demonstrate every potential benefit that practitioners might see for each intervention. My hope is that this book helps provide a fluid set of recommendations that are subject to change based on the available research. There is controversy in the subject of thyroid care; therefore, there are times where a provider may need to use clinical judgment to supersede the overarching recommendations and use innovative interventions, though keeping a patient's safety in mind is of primary importance in any of these decisions.

The main premise I wish to urge is that we focus on patient-centered care—listening closely to our patients, taking time and giving the attention needed to find the best course of action for each patient, one thyroid at a time.

Nicole Anziani, MS, RD, CDN, CDCES
Brooklyn, New York

Acknowledgments

I dedicate this work to my parents.

I wish to thank:

Debbie—my mother and the only person in my family to buy a book about thyroid when I was struggling, who called me to say, "I'm on page 108. I know all about you now. Oy … the hormones!" In 2015, while I was writing this book, my mother had a stroke. Much of this manuscript was written in her hospital room over a period of months. She constantly encouraged me to write, even when I was her caretaker. Thank you, *Bella*, for calling me your author.

Raul, my father and first editor—you've always encouraged me and believed in my abilities more than I did. You've shared in every triumph and struggle in my life. You were the first person I saw when I opened my eyes after surgery and the only one who offered to make me soup. I have endless appreciation for you and hope this makes you proud.

Grandma Mickey—along with my parents, you jumped around your kitchen doorway when the proposal for this book was approved. Thank you for being.

My paternal grandma, Elba Anziani, *Mamita*—you watched from the spirit realm as I wrote each word.

My brother Reuben "Madhu"—for providing me with healing (and a BioMat to write on) when my stress level was high.

My cousins J. Javier Anziani, JD, and Andre Anziani, JD, LLM, who provided contract expertise and advice.

Vanessa Alvarez, MS, RD—one of the best dietitians (and people) I know, whose empathy is unparalleled. You are part of the reason I am a dietitian today. Love you, my sister.

Jamillah Hoy Rosas, MPH, RD, CDCES—you were my first registered dietitian nutritionist boss and advisor in academic research at New York University. You always had faith in me, offering so many professional opportunities throughout my career. I will always look up to you as a mentor in the field.

Heather Perillo, MS, RD, CDN—you were a prime example of what a dietitian should aspire to be, first as my preceptor and later my colleague. Your training shaped my work and I thank you for having been my reference all these years.

All other instrumental mentors or dietitians in my academic life—including, but not limited to: Robert Fabini; Joan Cone; Christopher Dolder; Joan Thompson, PhD, MPH, RD, CDCES; Mary Mead, MEd, RD, CDCES; Nancy Hudson, PhD (Hon), MS, RD; Domingo Piñero, PhD; Antionette Franklin, MS, RD, CDN; Lisa Garback Garner, MS, RD, CDN; Mary Ellen Kelly, MS, RDN, CSSD, LDN; Pamela Purcell, MS, RD, CNSC, LDN; Eileen Ostrander, MS, RD, CDN; Cynthia Floyd, MS, RD, CDN, CDCES; Valerie L. Thomas, MS, RD, CDN; and Cindy Sizemore, MS, RD, LD.

The reviewers of this publication—thank you so much for your meticulous work and valuable comments that have made this publication a quality one.

Francesco Celi, MD, MHSc—your time and expertise as medical reviewer of this work is tremendously appreciated. Thank you for your contribution to thyroid research and for making this project stronger in so many ways.

The Academy of Nutrition and Dietetics—for taking on this project, and for your willingness to bring controversial information to light for the benefit of health professionals and patients alike.

Arlene Bregman, DrPh—for your organization of the local ThyCa meetings in NYC.

Yan Qi, PhD, and Liza Wong—for your suggestions and sharing conference notes.

Jaime Schehr, ND, RD—for pointing me to Dietitians in Integrative and Functional Medicine and the Natural Medicines Database for evidence-based research on dietary supplements.

Jennifer Trevillian and April Conlon, PharmD—for sharing resources and your personal journeys with me.

Anne Eller, PhD—for helping me understand the review process and sitting with me as I read the first set of reviews. Thanks for your expert insight!

Mally—you were one of the first to congratulate me on this project and sent motivational raps for the writing process. Thank you!

The B-School focus group who helped inspire the proposal for this book—Christine Gutierrez, LMHC; Tatiana Dellepiane; Dominick Quartuccio; Bertina Lee; Jennifer Spivak; Michelle Shemilt; and the memory of Jamie Lauren Zimmerman.

My friends and former coworkers at 185th Street, who made me feel like a rock star when they found out about this book—namely, Kimone Gilphilin, MS, RN; Lisa Calandra, DPM; Marlene Velez; and Danny Vairo.

All my colleagues at Cecelia Health—for your daily support.

The doctors who have helped me in my personal thyroid experience—Marc A. Cohen, MD, MPH; Arthur Spokojny, MD (RIP), and his medical staff (Sherine Smith); Naina Marballi, BASM; Alla Khalfin, DO; Maurice Beer, MD; Robin Berzin, MD; Lilli Link, MD; and Rekha Kumar, MD, MS. Each of you, in your own way, saved my life. How can I possibly thank you?

The most inspirational thought leaders I've encountered on my personal journey with thyroid cancer—Kris Carr; Terri Cole, LCSW; Sara Gottfried, MD; and Alan Christianson, ND—your work and vision have all contributed to my work. You've given me a reason to get up in the morning, permission to live without fear, and a fire to learn and hope for a better future.

Jancarlo—at the start of this project, your work ethic inspired mine; as the project continued, your laughter medicine became just as vital as my thyroid medicine. Thank you for your partnership and love.

All the souls I've had the privilege to work with, share time with, and learn from, and my community of friends and family—thank you for encouraging my medical and writing journeys. Your support has kept me truly alive.

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Nicole Anziani, MS, RD, CDN, CDCES, received her bachelor's degree in nutrition and dietetics from the University of California at Berkeley and completed her dietetic internship at the James J. Peters VA Medical Center in the Bronx, NY, coordinated with a master of science in clinical dietetics from New York University. She is a registered dietitian licensed in New York, a certified diabetes care and education specialist, and certified as a personal trainer, group fitness instructor, Ayurvedic wellness counselor, and reiki practitioner. As a thyroid cancer survivor, Anziani's passion and mission is to make nutrition care information for thyroid conditions easily accessible to practitioners and patients. Born and raised in Richmond, CA, Anziani now lives and works remotely via Brooklyn, NY.

Introduction and Medical Review

Francesco S. Celi, MD, MHSc, is the William G. Blackard Professor of Medicine and Chair of the Division of Endocrinology Diabetes and Metabolism at Virginia Commonwealth University in Richmond. Prior to joining the faculty of Virginia Commonwealth University, Celi worked for 10 years as clinical investigator at the National Institute of Diabetes, Digestive, and Kidney Diseases in Bethesda, MD. Celi is a graduate of the University of Rome "La Sapienza," and a diplomate of the Board of Internal Medicine, with subspecialty in endocrinology, diabetes, and metabolism. He was awarded the master's in health sciences by Duke University in Durham, NC. Celi conducts both clinical and translational research and his scientific interest is focused on the physiology and pathophysiology of thyroid hormone action as it relates to energy metabolism. Another area of research is in the mechanisms of adipose tissue differentiation and on the role of hormonal signaling (including thyroid hormone) on promoting differentiation of adipose tissue depots in thermogenic fat and its ability to dissipate energy. Celi has published more than 80 peer-reviewed manuscripts and has been invited to lecture at various institutions across the United States and Europe. Celi's clinical interests are thyroid disease, management of thyroid cancer, and treatment of diabetes.

Special Note

The Academy of Nutrition and Dietetics has adopted the increasingly common practice of dropping possessive apostrophes from the names of diseases and disorders. In alignment with the Endocrine Society, the American Medical Association, the 32nd edition of *Dorland's Illustrated Medical Dictionary*, and new material from National Institutes of Health, the *Health Professional's Guide to Nutrition Management of Thyroid Disease* spells terms such as Graves disease without an apostrophe. Other organizations and older publications may still maintain an apostrophe, but there is no difference in meaning between Graves disease and Graves' disease or Hashimoto thyroiditis and Hashimoto's thyroiditis.

Introduction

Francesco S. Celi, MD, MHSc

The action of thyroid hormone affects virtually all tissues of an organism, and it plays a critical role in the modulation of energy metabolism. Indeed, the dramatic effects of overt thyroid disease—particularly hyperthyroidism—on body weight and composition are valuable examples of the critical role hormones play in the regulation of metabolism. Aside from energy metabolism, thyroid hormone action affects other critical systems in an organism: first and foremost, the central nervous system, both during development and throughout adult life. Thyroid hormone action is also particularly important for bone growth and remodeling, as well for skeletal muscle development and function. Collectively, the action of thyroid hormone is pervasive, and its regulation is crucial for maintenance of body functions.

Disorders affecting the thyroid gland—both function and tumors—are very common in the general population and are disproportionally frequent in women. Although the symptomatology associated with overt thyroid dysfunction is quite characteristic and easily recognizable, most patients with thyroid disorders are affected by mild forms of thyroid dysfunction, with symptoms and signs that are often vague and aspecific. As individuals age, the prevalence of thyroid dysfunction can be as high as 12%, and population studies indicate that thyroid nodules are also very common. Increasing availability of sophisticated diagnostic tools and more widespread use of thyroid ultrasound has contributed to the increased frequency of diagnoses of thyroid dysfunction, nodules, and cancer in recent years.

This increase in diagnoses of thyroid pathology has been mirrored by an increase in awareness among the general public of the role (whether real or perceived) that thyroid function plays regarding an individual's sense of well-being. This has progressed to the point that assessment of thyroid function is one of the first steps providers undertake while evaluating symptomatology that is not clear and that may have overlap with other chronic conditions. As a consequence, thyroid disease and dysfunction are very common concerns among patients and providers, and, quite often, both parties share the notion that subclincal or unrecognized thyroid dysfunction may be the root cause of otherwise unexplained symptoms. As a consequence, modulation of thyroid function, via either therapy or dietary supplements, is often employed to address a variety of conditions, particularly those involving weight gain, low energy, chronic fatigue, and depression.

Against this background of increased awareness and, in some cases, perhaps excessive expectations, it's important to consider that the thyroid gland and its function is heavily affected by nutrition and adequate delivery of oligo-elements: in particular, iodine and selenium. In fact, iodine deficiency represents the most common preventable cause of intellectual disabilities worldwide. Although the United States is considered an iodine-sufficient area, some conditions and dietary habits can significantly impact iodine intake, resulting in iodine deficiency. Thyroid pathology itself, in either the acute or recovery phase of disease,

often requires nutritional intervention to optimize the healing process and to prevent additional morbidity.

Some lay literature and a substantial number of practitioners—particularly in the fields of alternative and complementary medicine—strongly support using nutritional supplements and modification of diet to optimize thyroid function, either by addressing nutritional deficiencies or stimulating immune function. Although the empirical evidence of this in humans is marginal and of limited quality, it is important to note that *in vitro* experiments and mechanistic studies carried out in laboratory animal or cell culture systems have indicated that nutrition and oligo-element supplementation play roles in the pathophysiology of the thyroid. While this is important, it's also important to recognize the knowledge gap between findings in experimental models and clinical relevance in patients.

The *Health Professional's Guide to Nutrition Management of Thyroid Disease* takes a unique course by defining the role of nutrition and dietary supplementation in thyroid physiology and pathology. By systematically defining the various physiologic states and pathologic conditions affecting the thyroid, this book provides relevant information on the potential roles of nutrition intervention and use of dietary supplements. It strives to define the mechanistic rationale for such interventions; whenever possible, it provides references to relevant human studies. It provides references to guidelines of leading professional organizations that are dedicated to the study and care of thyroid disease, and it recognizes the gaps in knowledge of the effects of nutritional interventions and dietary supplementation that exist in relation to thyroid disorders.

This book's systematic approach to the topic and its extensive references to high-quality published research make it a useful reference for nutritionists and practitioners alike. At the same time, each chapter's introduction provides a good framework of the problem presented, which enables a nonmedical audience to better understand the roles nutrition and dietary supplementation can serve in the amelioration of thyroid disease, all while maintaining realistic expectations. Finally, the extensive references and resources herein provide a comprehensive tool set to help readers further deepen their knowledge of the interactions among nutrition, dietary supplementation, and thyroid function.

SECTION 1

Introduction to Thyroid Disorders

1

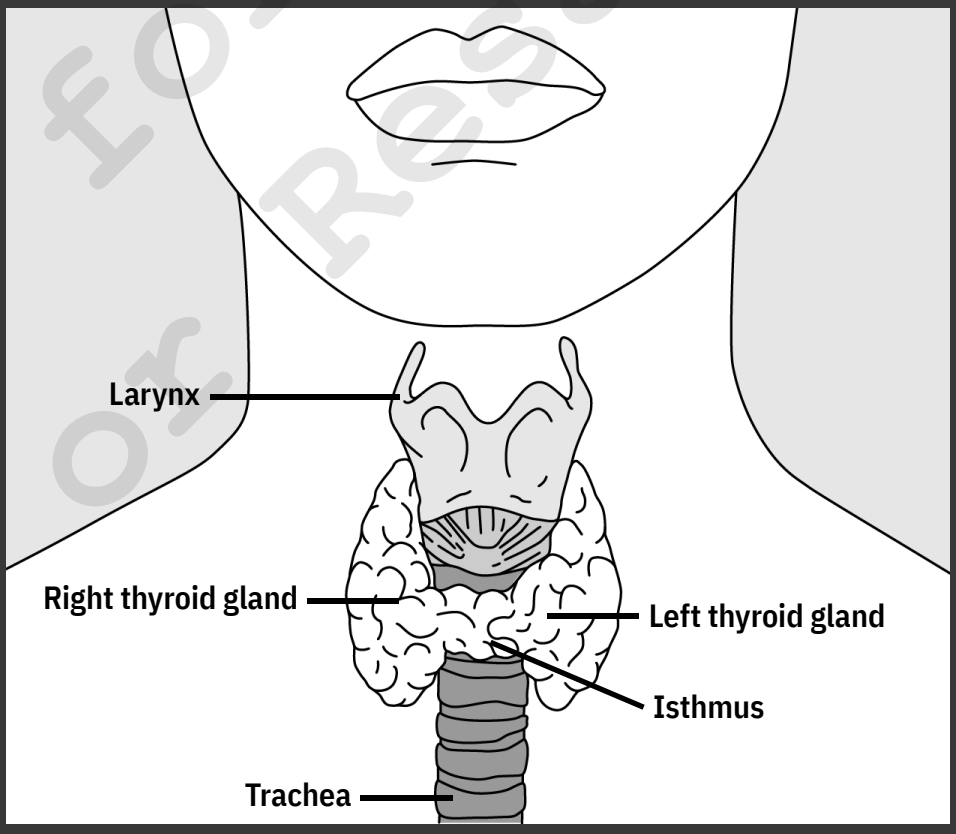
Overview of the Thyroid and Its Function

INTRODUCTION

The thyroid is a butterfly-shaped endocrine gland weighing less than 1 ounce (28 g) in healthy adults and children. It is located in the middle of the lower neck, in front of the trachea (see Figure 1.1).[1] Thyroid hormones regulate the body's metabolism and affect nearly every action of every cell, including such vital functions as heart rate and energy level.[2,3]

The thyroid works in conjunction with the pituitary gland and the hypothalamus. The hypothalamus, located in the lower central area of the brain, produces thyrotropin-releasing hormone (TRH) in response to external factors and stressors.[4] TRH travels to the pituitary gland at the base of the brain. There, thyrotropin/thyroid-stimulating hormone (TSH) is produced. TSH is sent to the thyroid gland to regulate thyroid hormone storage, production, and release. Figure 1.2 illustrates the feedback mechanisms of the hypothalamic-pituitary-thyroid (HPT) axis.

FIGURE 1.1
THYROID ANATOMY



5 Signs and Symptoms, Risk Factors, and Etiology of Hypothyroidism

INTRODUCTION AND BACKGROUND

Hypothyroidism, or underactive thyroid, is the most common thyroid disorder. It is thought to affect approximately 4.6% of the US population over age 12,[1] and that estimate increases to as high as 10% outside of the United States.[2] In hypothyroidism, the thyroid gland does not produce enough thyroid hormones, including triiodothyronine (T3) and thyroxine (T4), to maintain the body's functions. This decreases metabolism, including cell activity and regeneration, and may be related to unintentional weight gain.[3] Refer to Chapter 2 for more information regarding thyroid disease and weight regulation.

Hypothyroidism can increase cardiovascular risks and can cause mood changes, fatigue, and gastrointestinal distress, all of which can be barriers to healthy lifestyle changes. The health or nutrition professional is challenged with the task of helping patients set realistic goals for diet and exercise while taking into account possible micronutrient deficiencies and interactions between thyroid medications, foods, and supplements. It is important to coordinate with the patient's medical team in order to promote improved outcomes.[4]

SIGNS AND SYMPTOMS

There are many potential physical and mental symptoms of hypothyroidism. Not all symptoms are present in every patient, and some patients may not exhibit any symptoms; some can also be subtle, especially in subclinical hypothyroidism (SCH).[5] It is important to note that many hypothyroid symptoms overlap with those seen in other chronic conditions. Box 5.1 summarizes some common signs and symptoms of hypothyroidism.

BOX 5.1 SIGNS AND SYMPTOMS OF HYPOTHYROIDISM [5-9]

Physical Symptoms of Hypothyroidism

- Brittle nails
- Chronic sinusitis
- Constipation
- Decreased libido
- Decreased perspiration
- Difficulty losing weight
- Dry and coarse skin
- Dry and gritty eyes
- Enlarged neck
- Fatigue
- Goiter
- Hair loss
- Hearing loss
- Heavy and irregular menses
- Hoarse voice or voice changes
- Infertility
- Insomnia
- Intolerance to cold
- Joint pain
- Mood swings
- Muscle cramps
- Puffiness of hands and face
- Slowed ankle-reflex relaxation time
- Slowed speech
- Small thyroid gland
- Tinnitus
- Weight gain

continued on next page

BOX 5.1 SIGNS AND SYMPTOMS OF HYPOTHYROIDISM [5-9] (continued)

Mental Symptoms of Hypothyroidism

Brain fog
Confusion
Depression
Difficulty thinking and concentrating
Irritability
Memory impairment
Mental apathy

Symptoms of Severe Hypothyroidism

Carpal tunnel syndrome
Elevated lipids
Hyponatremia can arise within several weeks of onset
Pituitary hyperplasia with or without hyperprolactinemia and galactorrhea
Serum creative kinase and other muscle/hepatic enzymes may be abnormal
Sleep apnea

RISK FACTORS

The following factors may predispose a person to hypothyroidism:

- **Personal history of a thyroid problem**, such as goiter or nodules.[1]
- **Personal history of postpartum thyroiditis or autoimmune thyroid disease** (eg, Hashimoto thyroiditis*).
- **Family history of thyroid disease**, which increases the risk of developing autoimmune thyroid disease. Studies have shown that up to 60% of first-degree relatives of people with autoimmune thyroid disease also have thyroid antibodies, which may signal future development of an autoimmune thyroid condition.[10]
- **Being Female**; women are five to eight times likelier than men to develop hypothyroidism.[8] Women with positive antibodies and high thyroid-stimulating hormone (TSH) levels have a higher annual risk (4%) of developing hypothyroidism than those with either factor alone (2% to 3%).[5]
- **Age**; hypothyroidism is more common over the age of 60.[1] As many as one in four patients in long-term care may have undiagnosed hypothyroidism.[11]
- **Autoimmune diseases and endocrine disorders**, both in personal or family history, such as lupus, Sjögren syndrome, pernicious anemia, type 1 diabetes, celiac disease, rheumatoid arthritis, Addison disease, or polycystic ovary syndrome.[1]
- **Turner syndrome**, a genetic disorder in females.[1]
- **Being pregnant or up to 6 months postpartum**[1]; additionally, other hormonal imbalances and menopause can initiate thyroid imbalances.
- **Receiving radiation to the thyroid, neck, or chest**.[1]

ETIOLOGY

There are several causes of hypothyroidism, which can be categorized as biological or nonbiological causes.

Biological Causes of Hypothyroidism

Hashimoto disease, otherwise known as chronic lymphocytic thyroiditis, autoimmune thyroiditis, or Hashimoto thyroiditis, is an autoimmune disorder[12] that is the most common cause of hypothyroidism in the United States.[1] Hashimoto disease, which may cause more than half of hypothyroidism cases in North America, is most common in women between 30 and 50 years of age.[12]

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Inflammation of the thyroid gland from an autoimmune attack can lead to excess thyroid hormone production and a hyperthyroid state[13] known as hashitoxicosis. This inflammation can lead to destruction of the thyroid gland and a gradual inability to produce thyroid hormones. As this develops, the pituitary gland releases TSH in increasing amounts to signal the thyroid to produce more hormones. In some cases, this may lead to a goiter or an enlarged thyroid gland.[9]

Experts suggest that this may be triggered by a buildup of environmental toxins in the thyroid, intestinal permeability, gluten and casein in the diet, drops in blood glucose, stress, and other infections or inflammation causing stimulation of T-helper cells or lymphocytes (Th1 or Th2). Practitioners have found that treating these root causes may halt the disease process,[9] but experimental evidence is needed.

Thyroiditis is inflammation of the thyroid gland that is often caused by autoimmunity or infection. In some cases, hormone secretion is increased, leading to 1 or 2 months of thyrotoxicosis.[12] The damage caused from the ensuing inflammation can lead to hypothyroidism[1] or can resolve spontaneously.

Reidel thyroiditis is a rare, chronic inflammatory disease of the thyroid gland in which dense fibrosis replaces normal thyroid tissue. It may permanently damage the thyroid, causing hypothyroidism in 30% of cases.[14] In de Quervain thyroiditis, the thyroid gland rapidly swells and is painful and tender.[15] The patient experiences no pain in instances of silent thyroiditis.[16]

Ord disease is a type of autoimmune thyroid disease that does not involve development of a goiter. Some researchers consider it to be part of the same disorder as Hashimoto disease.[17]

Postpartum thyroiditis, discussed in more detail in Chapter 17, has three possible outcomes. It may cause transient thyrotoxicosis in the mother, which resolves. It may cause transient hypothyroidism following transient thyrotoxicosis in the mother, and both subsequently resolve. Last, it may develop into a type of Hashimoto thyroiditis in which hypothyroidism in the postpartum period progresses into overt hypothyroidism.[18]

Genetic causes; several genes affect normal fetal thyroid development,[19] with some decreasing even a normal thyroid's ability to produce thyroid hormones.[20] Congenital hypothyroidism, in which a child is born with partial or total loss of thyroid function, occurs when there are mutations in the DUOX2, PAX8, SLC5A5, TG, TPO, TSHB, and TSHR genes. This affects one in every 3,000 to 4,000 newborns, is inherited 15% to 20% of the time,[21] and can lead to intellectual disabilities and growth failure. For this reason, most newborns in the United States are screened for hypothyroidism, as early treatment can prevent complications (this is discussed in further detail in Chapter 17).[1] The vast majority of cases of congenital hypothyroidism have unknown genetic causes.

Pituitary and hypothalamic disorders; normally, the hypothalamus stimulates the pituitary gland to make TSH. Malfunctions in these mechanisms can result in hypothyroidism.[22] It is believed that as many as 1 in 10 adults worldwide has an adenoma, or benign tumor of the pituitary gland, yet many of these tumors do not cause harm or secrete hormones (those with no clinical impact are commonly referred to as incidentalomas). Rarely, pituitary

choose to use a compounding pharmacy, the pharmacy should meet stringent quality standards, such as holding a sterile compounding pharmacy license.

How to Dose Medications

When a health professional initiates thyroid replacement therapy, the ATA recommends that a "patient's weight, lean body mass, pregnancy status, etiology of hypothyroidism, degree of TSH elevation, age, and general clinical context, including the presence of cardiac disease," along with the TSH goal, should be taken into account. Patients with a goal of TSH suppression generally require more thyroid hormone replacement; those whose thyroids have been completely removed generally need more than Hashimoto patients. Patients who have received radioactive iodine ablation for Graves disease may need varying amounts depending on residual thyroid function post-treatment. Patients who have experienced a significant change in weight, who are aging, who are pregnant, or who have changed their thyroid dosage should have blood work reassessed in 4 to 6 weeks.[6] Table 6.2 includes initiation and dosing recommendations for several subsets of patients.

TABLE 6.2 THYROID CONDITIONS AND MEDICATION DOSING [1,6]

Patient Profile	L-T4 Dose (per kg body weight)	Thyroid-Stimulating Hormone Goal	How to Initiate and Progress Therapy
Minimal endogenous thyroid function in middle-aged patients	1.6-1.8 µg/kg actual body weight 2.0-2.1 µg/kg for some patients Some use ideal vs actual body weight	0.5-2.0 mIU/L for good balance	Initiate at full dose
Thyroid cancer patients	2.1-2.7 µg/kg	Thyroid-stimulating hormone (TSH) suppression, or individualized to address prognosis/type of cancer	Initiate at full dose
Mild hypothyroidism: serum TSH ≤ 10 mIU/L in young and middle-aged patients	–	0.5-2.0 mIU/L for good balance	Initiate at full dose, usually 25-75 µg/d (not weight-based therapy)
Elderly patients over age 65 (less lean body mass, less hormone need)	–	Higher serum TSH goal range 4-6 mIU/L in those >70 years of age	Start low and go slow: generally initiate at 50 µg, and adjust by 12.5-25 µg/d up or down depending on TSH
Cardiac patients with coronary artery disease	–	0.5-3.5 or 4 mIU/L, per American Thyroid Association	Start low and go slow: gradual increases based on TSH and symptoms, with adjustments of 12.5-25 µg/d
Patients who are severely hypothyroid after treatment for hyperthyroidism	Variable	0.5-3.5 or 4 mIU/L	Initiate full dose

of antidepressants to changes in the brain's metabolism of thyroid hormones, suggesting that the use of liothyronine (L-T3) in conjunction with antidepressants may be beneficial.[57] This has been further studied and confirmed in human studies.[58,59]

Low thyroid hormone levels can cause residual symptoms that linger post-treatment.[60] Graves disease (GD) patients have been shown to have a lower quality of life up to 21 years following treatment for hyperthyroidism,[61] and research has shown that 35.6% of patients with GD who have had normalized thyroid levels for over 6 months still experience psychological distress and anxiety, and that 95.6% of them were depressed.[62] Prevention and education regarding early warning signs, such as changes in sleep, appetite, energy, self-esteem, motivation, concentration, or sexual interest, are recommended.

Nutrition and Lifestyle Approaches to Improve Depression and Mood

Researchers have examined the benefits of several nutrients, supplements, and lifestyle behaviors and their effects on depression. For example, research has shown that increasing n-3 fatty acid consumption helps alleviate depression,[63] as does stress management and development of regular sleep patterns.[64] Nutrient interventions include consuming foods rich in beta-carotene; vitamin C; vitamin E; lean proteins; and complex vs simple carbs, as well as considering a Mediterranean diet overall.[64] St John's wort has been used as a supplement to manage depression, but it is important to keep in mind that these supplemental therapies should be administered under qualified medical supervision.[64] Most interventions are not recommended in pregnant or lactating women, and practitioners should exercise caution when working with patients with certain medical conditions or patients taking additional medications. If the RDN or other health care provider wishes to recommend complementary or alternative therapies, more thorough investigation into side effects, food–drug and herb–drug interactions, and contraindications is advised and should be individualized to the patient with the medical team.

FIBROMYALGIA

Fibromyalgia, a chronic and disabling condition characterized by pain, fatigue, stiffness, and multiple tender points, afflicts 2% to 4% of the North American population.[65] It is thought that central nervous system dysfunction and lifestyle factors, such as sleep difficulties, stress, infection, injury, nervous system changes, changes in muscle metabolism, and a family history, contribute to its development.[66]

In fibromyalgia, a constant low-level activation of the coagulation system has been observed in limited research.[67] This creates a soluble fibrin monomer (SFM) that coats the inside of blood vessels, limiting oxygen and nutrients from entering the cells. Decreased oxygen may lead to pain, fatigue, and brain fog. SFM development can lead to bacterial growth and infection. These effects can make thyroid hormone less efficient[68]; however, it is important to note that this is a speculative theory.

Some alternative medicine practitioners believe that people with fibromyalgia can be cured or improved by treating the individual with high doses of triiodothyronine (T3) along with nutritional supplements, diet, and exercise.[69,70] This protocol can be dangerous, however, and is not accepted as a treatment practice. The high T3 dose utilized in one study may induce hyperthyroidism, but a lower dose could still provide benefits with a decreased risk of iatrogenic hyperthyroidism. Fibromyalgia has some links to thyroid disease, including the following:

- One out of every three fibromyalgia patients also has autoimmune thyroid disease.[71]

SECTION 6

Appendixes

APPENDIX E

Sample Food Plans (Low-Glycemic, Gluten-Free, Anti-Inflammatory, Autoimmune Paleo)

LOW-GLYCEMIC SAMPLE 3-DAY DIET

There are different methods for designing a low-glycemic food plan. The sample 3-day menus for the low-glycemic diet provided here contain:

- Unlimited carbohydrates from low-glycemic vegetables, such as broccoli, asparagus, spinach, chard, kale, cabbage, bok choy, arugula, sea vegetables, and so on. (Note: many of these are goitrogens, so cooking would be preferable to consuming raw.)
- Up to ½ cup of whole grains, such as brown, black, and red rice; quinoa; amaranth; buckwheat; or teff, or up to ½ cup of starchy vegetables such as winter squash, peas, potatoes, corn, and root vegetables, per day.
- Up to ½ cup of legumes, such as lentils, chickpeas, split peas, edamame, black beans, or navy beans per day.
- Up to ½ cup of berries, such as blueberries, cherries, blackberries, and raspberries, per day and/or one to two pieces of apple, pear, or stone fruit, such as plum, peach, or nectarine.
- From 30 to 50 grams of fiber per day.

	BREAKFAST	LUNCH	SNACK	DINNER
DAY 1	Blended shake made with pea protein powder, ½ cup frozen berries, 1 cup unsweetened almond milk, 1 tablespoon flaxseed oil, 1 handful spinach, and 2 teaspoons almond butter	Salad made with ½ can rinsed white beans served over a bed of arugula and topped with 1 small can artichoke hearts; drizzle with extra virgin olive oil and balsamic vinegar and sprinkle with black pepper	1 ounce almonds	4 ounces salmon filet served with ½ cup sweet potato and steamed asparagus drizzled with 2 teaspoons olive oil
DAY 2	2 hard-boiled eggs served on a bed of arugula with ½ cup raspberries on the side	Smoked salmon served with sliced Kirby cucumbers, tomatoes, kalamata olives, and hummus over a bed of romaine lettuce	Turkey slices rolled up in lettuce leaves	Quick sauté over medium heat: ½ cup precooked brown rice; prewashed spinach and kale; 2 teaspoons olive oil; chopped garlic; and 4 ounces canned chicken
DAY 3	Egg-white omelet made with spinach, ¼ avocado, and sliced tomato, served with ½ cup blueberries	½ cup cooked lentil soup with a side salad, ½ avocado, and sliced tomatoes and ½ sliced peach	Celery with almond butter	Chopped tofu and bok choy sautéed in tamari sauce and served over ½ cup quinoa

Continuing Professional Education

This edition of *The Health Professional's Guide to Management of Thyroid Disease* offers readers 8 hours of Continuing Professional Education (CPE) credit. Readers may earn credit by completing the interactive online quiz at the following website: https://publications.webauthor.com/health-pro-guide-thyroid

Index

Page numbers followed by *f* indicate figures, page numbers followed by *t* indicate tables, and page numbers followed by *b* indicate boxes.

Errata

The Health Professional's Guide to Nutrition Management of Thyroid Disease

A corrupt file caused several symbol replacement errors in section 1.

Page and location	Original	Corrected
4, Understanding Diagnostic Criteria	The statement noted that in the future, it is likely that the "limit of the serum TSH euthyroid reference range will be reduced to 2.5 mIU/L because less than 95% of rigorously screened normal euthyroid volunteers have serum TSH values between 0.4 and 2.5 mIU/L. … A serum TSH result between 0.5 and 2.0 mIU/L is generally considered the therapeutic target for a standard levothyroxine (L-T4) replacement dose for primary hypothyroidism."[10]	The statement noted that in the future, it is likely that the "limit of the serum TSH euthyroid reference range will be reduced to 2.5 mIU/L because **more** than 95% of rigorously screened normal euthyroid volunteers have serum TSH values between 0.4 and 2.5 mIU/L. … A serum TSH result between 0.5 and 2.0 mIU/L is generally considered the therapeutic target for a standard levothyroxine (L-T4) replacement dose for primary hypothyroidism."[10]
7, Box 1.3	**Free Thyroxine, Free T4 (FT4)** The amount of unbound T4 in the blood; >1% of T4 is unattached. Free thyroxine affects tissue function in the body. Most tests are affected by the carrier protein concentration in the serum.	**Free Thyroxine, Free T4 (FT4)** The amount of unbound T4 in the blood; **<**1% of T4 is unattached. Free thyroxine affects tissue function in the body. Most tests are affected by the carrier protein concentration in the serum.
7, Box 1.3	**Free Triiodothyronine, Free T3 (FT3)** The amount of free, unbound T3 in the blood; >1% of T3 is unattached. T3 has a greater effect on the body's utilization of energy than T4.	**Free Triiodothyronine, Free T3 (FT3)** The amount of free, unbound T3 in the blood; **<**1% of T3 is unattached. T3 has a greater effect on the body's utilization of energy than T4.

Page 18 ▪ Under the heading Thyroid-Specific Weight Loss Techniques, in the list of biomarkers commonly tested, number 2 should read **Thyroxine** (T4) (free or total).

Page 115 ▪ In Box 11.2, the first sentence in the box should read as follows: Thyroid antibodies (positive thyroid stimulating immunoglobulin [TSI] indicating Graves disease or, if thyroid peroxidase [TPO] but not TSI or **thyroid-stimulating hormone receptor antibodies** [TRAbs], Hashimoto thyroiditis).

Page 140 ▪ The title of Box 14.1 should be Risk Stratification of **Recurrence of** Thyroid Cancer.

Please contact publications@eatright.org with any questions or concerns.

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September 2020